the collaborative process (not the outcomes of the scientific research or COTC translation and dissemination activities) by assessing the perceptions of the collaboration by advocates and scientists who responded to our survey and were interviewed. This overview of the centers demonstrated, as noted by Wolff and Barlow, that the centers varied with regard to their experience with community-based participatory research. Wolff and Barlow note in their letter that the Bay Area COTC was the one center that incorporated the principles of community-based research and, based on our research, provided the best example of successful advocate–scientist collaboration among the BCERCs. Therefore, throughout our article (Baralt and McCormick 2010) we used the Bay Area BCERC (as well as in the Supplemental Material, in which we elaborated on a number of recommendations for future breast cancer–environment research collaborations) as a model for future collaborative projects. Additionally, we were careful in noting the limitations of our methods, acknowledging that our findings reflect only our sample of possible respondents and therefore may not be generalizable to all center advocates and scientists.

Our article (Baralt and McCormick 2010) and recommendations are both in the spirit of furthering the work of the BCERCs and projects like the BCERCs that engage in the "ongoing, interactive, collaborative, critical process of science and advocacy," as Wolff and Barlow describe their work with the BCERCs over the past 7 years. We encourage other researchers to continue investigating how environmental breast cancer research and other types of participatory projects can best serve the interests of science, advocates and policy-makers.

The authors declare they have no actual or potential competing financial interests.

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Redefining Low Lead Levels

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In the January 2011 issue of *EHP*, Giddabasappa et al. (2011) reported that gestational lead exposure (GLE) of C57BL/6 mice produced selective nonmonotonic increases in the numbers of rods and cone bipolar cells (BCs) in the adult retina. Interestingly, this increase was characterized by an inverted U-shaped dose–response curve. Moreover, findings of this study showed that GLE increases and prolongs proliferation of retinal progenitor cells (RPCs) without decreasing apoptosis. Consequently, this phenomenon produced an adult retina with normal lamination and a selectively increased number of rods and BCs. These results should be considered to define a more adequate risk assessment at low levels of lead exposure. In fact, other published articles have indicated that lead induced a biphasic dose–response relationship (Calabrese and Baldwin 2003).

In experiments in Swiss mice using low-level lead exposures similar to and lower than those used by Giddabasappa et al. (2011), we observed an increase in the number of red blood cells, in female gestational parameters, and in Th1 cytokine levels (Iavicoli et al. 2003, 2004, 2006a, 2006b). For this reason, it would be interesting if Giddabasappa et al. could verify this increase in the number of neurons in the rod-signaling pathway at even lower blood lead levels (< 10 μg/dL). The findings of our studies were also implicated over several generations.

In any case, we agree with Giddabasappa et al. (2011) that their findings, as ours, raise complex issue for toxicologists, pediatricians, public health regulators, and risk assessors who need to incorporate the occurrence of such U-shaped dose responses in the hazard and risk assessment process. In this context, these findings could be explained by the hormesis phenomenon, which is a dose–response relationship characterized by low-dose stimulation and high-dose inhibition (Calabrese 2008, 2009).

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Editor's note: In accordance with journal policy, Giddabasappa et al. were asked whether they wanted to respond to this letter, but they chose not to do so.